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Election to the R.C.V.S. Council

ONCE again the profession is faced with its annual duty of electing its representatives to the Council of the Royal College of Veterinary Surgeons. In many ways this annual election is one of the most important events in the veterinary calendar, and it causes some disquiet that many members appear to regard it so casually that they do not trouble to register their votes. Our small profession faces many opportunities in a world of expanding knowledge and new techniques, equally these opportunities are coupled with difficult problems of policy and co-operation with outside bodies, who may not share our views or agree with our conclusions. In such circumstances it should hardly need arguing that the Council of the Royal College should represent the profession to the fullest degree possible, and this can only be achieved by a substantial majority of members recording their votes in the annual election. We must remember that since the 1948 Act the Council comprises a substantial proportion of persons nominated by the Universities, the Privy Council, and the Republic of Ireland. In fact there are 17 nominated members, 20 generally elected members and two elected by members resident in Eire. There can be no question that the nominated members, most of whom are veterinary surgeons, bring a breadth of experience and wisdom which have immeasurably strengthened the Council. Furthermore, their views largely reflect those of the profession as a whole; nevertheless, they are not elected and it is essential that those representatives who are elected should be elected on a substantial vote.

Some veterinary surgeons have stated in these columns and elsewhere that they cannot vote in favour of candidates of whom they know little or nothing. To try and help overcome such objections we publish in this issue a list of candidates accompanied by brief biographical details of their careers and services to the profession. It is to be hoped that this aid and a sense of duty on the part of each member will result in a poll more nearly representative of the main body of the profession.

The Toxicity of Rodenticides
II. Red Squill and Zinc Phosphide

BY

*R. J. FITZPATRICK, J. L. McGIRR and D. S. PAPWORTH
Weybridge

Red Squill

THE preparations of squill employed for rat poisoning are derived from the dried, ground bulbs of red variety of the sea onion, *Urginea maritima*, a plant indigenous to Mediterranean countries. The bulbs contain glycosides some of which have been isolated and which have a digitalis-like action on the heart (Stoll & Renz, 1942). According to the latter authors, red squill would appear to be the oldest rodenticide in use, it having been recommended by an Arabian author for the eradication of mice in the early 13th century. Another author (Shepard, 1951) maintains that references to red squill as a rodenticide have been made on stone tablets of the pre-Christian era.

The reputation of red squill as a " safe " rodenticide was based upon its extreme unpalatability to domestic livestock and the fact that when eaten it is vomited, thus preventing its absorption. The rat, which is said to be incapable of vomiting, is not protected in this manner. Despite its usefulness as a rodenticide, however, rats which have eaten the material and survived will show shyness towards the compound, and all the drawbacks of acquired bait shyness are therefore applicable to this material. There are also the disadvantages that it is an irritant compound to handle, has to be imported and is relatively expensive. The material is also liable to be very variable in toxicity to rodents.

The toxicity of red squill to domestic livestock has been investigated by Fitzpatrick (1952) and Table I* provides a summary of his results regarding toxic doses for various species.

Palatability trials by the same author show that under normal farm feeding conditions it is very unlikely that any pigs or ruminants would voluntarily eat sufficient bait material to prove harmful. For example, pigs even when starving will completely refuse not only bait of 10 per cent. strength normally used in the field, but even baits of 2 per cent. strength or less. Dogs and cats are as susceptible as other species, but they are very sensitive to both the odour and taste of red squill and this is an adequate safeguard against poisoning. Vomiting cannot be regarded as a reliable form of protection. Death after oral administration of red squill was noted in the pig (despite violent emesis), and in cats (in which emesis was usually absent). There is little danger from the ingestion of poisoned rats by cats and dogs, since the alimentary tract containing most of the squill is not eaten on these occasions. (Bureau of Animal Population, 1948.)

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The symptoms produced by this poison are often of the nature of a C.N.S. stimulation, the animals showing hyperaesthesia, inco-ordination of movements and frank convulsions. Frequently this is associated with intermittent periods of intense and general depression and in some cases—particularly in ruminants—only the depression is seen. Emesis and diarrhoea may occur but are not invariable. Symptoms usually develop within 12 hours and although they may continue for up to a week, the majority of deaths occur within three days of taking the material.

TABLE I
AMOUNT OF FIELD BAIT* IN OUNCES NECESSARY TO POISON SPECIFIED ANIMALS

Species	Body weight lb.	Body weight kg.	Toxic dose of squill powder mg. per kg.	Amount of bait
Pig	33	15	200	1 oz. or less
"	100	45·5	200	3 "
"	300	135	200	9 "
Bovine	1,000	455	250	38 "
"	100	45·5	100	1½ "
Sheep	100	45·5	250	4 "
Goat	100	45·5	500	7½ "
Dog	11	5	145	¼ "
"	25	11·5	145	½ "
"	100	45·5	145	2 "
Cat	4½	2	100	1/15 " (= 30 grains)
"	11	5	100	1/6 " (= 75 ")
Rabbit	4½	2	300	1/5 " (= 90 ")

* Bread and sausage rusk paste containing 10 per cent. w/w of a red squill powder with an LD/50 for male rats of 490 mg. per kg.

The most obvious lesions affect the alimentary canal and circulatory system. Gastritis and enteritis are usually marked with pronounced congestion, swelling and often ulceration of the mucosa. Frequently the contents of the stomach, small intestine and large intestine are extremely haemorrhagic. These alimentary lesions are, however, less severe in ruminants.

In all species there is usually congestion of the abdominal and thoracic organs. There is particularly marked congestion of the mesenteric vessels and the mesenteric glands are swollen, oedematous and congested, even in animals with only mild inflammatory changes in the intestine.

Kidney, liver, lungs and myocardium show signs of swelling and congestion, and histological examination shows general degenerative changes.

As can be seen from Table I, goats are relatively the most resistant animals, but except for very young animals it is unlikely that a toxic dose for normal livestock would be available after baits had been laid on a farm. Emesis will occur in some species—pigs, dogs and cats—after eating red squill, but does not always save the animal.

Zinc Phosphide

Zinc phosphide is a black powder, slightly soluble in water and possessing a garlic-like odour due to the presence of phosphine gas. This odour although repellent to man and domestic animals has been reported to attract rats.

Baits used for rat poisoning are generally made up of 2·5 per cent. by weight of the zinc phosphide to a mixture of damp sausage rusk, bread mash, soaked wheat or damp rolled oats. Five per cent. baits may also be employed.

The LD_{50} for wild *R. norvegicus* rats (Dicke & Richter) is of the same order as that reported for man and dogs (U.S. Pub. Hlth. Serv., 1949) at 40 mg. per kg.

Table II provides a summary of the toxicity trials of zinc phosphide carried out at Weybridge. One point worth noting is that the average lethal or clinical response

TABLE II

TOXIC EFFECTS OF ZINC PHOSPHIDE

Animal	Reference number	Weight, kg.	Dose, gm.	Dosage rate, gm. per kg.	Route	Effect	Survival time	Analytical data from gastro-intestinal tract contents p.p.m.		
Cow ...	1125	483	9·66	20	Capsules	Survived	—		—	
„ ...	1125	483	19·32	40	„	Died	72 hours	Abomasum	>100	+++
								Intestine	< 20	+
Goat ...	1396	61·5	2·456	40	In food	Survived	—		—	
„ ...	1396	61·5	3·684	60	Capsules	Died	4 weeks	1st stomach	20–100	++
								2nd „	< 20	+
								3rd „	< 5	—
								4th „	< 5	—
„ ...	1212	46	0·924	20	In food	Survived	—		—	
„ ...	1212	46	1·848	20	Capsule	Died	24 hours	1st stomach	20–100	++
								2nd „	>100	+++
								3rd „	>100	+++
								4th „	> 20	+
								Intestine	< 20	+
Sheep ...	2337	84	3·36	40	In food	Survived	—		—	
	2337	84	5·04	60	Capsule	Died	20 hours	1st stomach	>100	+++
								2nd „	>100	++++
								3rd „	>100	++++
								4th „	20–100	++
								Intestine	< 20	+
„ ...	2441	30	1·20	40	Capsule	„	21 „	1st stomach	< 20	+
								2nd „	>100	+++
								3rd „	20–100	++
								4th „	< 20	+
								Intestine	< 20	+
„ ...	2448	30	0·9	30	„	Survived (killed one week later)	—	1st stomach	20–100	++
								2nd „	>100	++++
								3rd „	>100	++++
								4th „	>100	+++
								Intestine	< 20	+
Pig ...	4686	35	2·1	60	Stomach tube	Died	8 hours	Stomach	20–100	++
								Intestine	>100	+++
„ ...	4719	77	3·08	40	„	„	36 „	Stomach	>100	+++
								Intestine (bloody liquid)	< 5	—
„ ...	4686	25	0·5	20	In food	Survived	—		—	
„ ...	4686	27	1·0	40	„	„	—		—	
„ ...	4686	31	1·5	50	„	„	—		—	
„ ...	4686	34	2·0	60	„	„	—	Only 50 per cent. food eaten		
„ ...	4727	41	0·818	20	„	„	—		—	
„ ...	4727	41	1·23	30	„	„	—		—	
„ ...	4727	76	2·28	30	Stomach tube	„	—		—	
„ ...	4719	41	0·818	20	In food	„	—		—	
„ ...	4719	41	1·23	30	„	„	—		—	

Guide key : — Negative test Colour yellow.
+ Slight positive „ greenish yellow.
++ Definite „ „ blue.
+++ Strong positive Colour dark blue.
++++ Very marked positive „ black blue.

doses are all of the same order, there is not the divergency commonly encountered among different species with some other poisons. The method used for the detection of zinc phosphide was that of Salt (1945), which consists of heating the material containing zinc phosphide with dilute hydrochloric acid and absorbing the liberated phosphine in potassium permanganate (Elmore & Roth, 1943). The phosphate thus formed is then converted into a reduced phosphomolybdic compound, according to the method of Fiske & Subbarrow (1925), and the excess potassium permanganate is decolorised by the reducing agent. The colour developed is proportional to the amount of phosphate formed from the original zinc phosphide, the following colour reference being noted :—

Pale yellow	...	No phosphide.
Greenish-blue	...	Less than 0·2 mg. Zn_3P_2.
Blue ...	...	0·2–1 mg. Zn_3P_2.
Intense blue	...	More than 1 mg. Zn_3P_2.

This method was used since results depending on the estimation of zinc are very unreliable in view of variations in zinc levels in the animal body and knowledge of these levels.

The symptoms of zinc phosphide poisoning revealed by these experiments were the general reactions to a toxaemia, nothing specific being apparent. There was usually a slight odour of phosphine apparent on opening the stomach, but this was more marked with pigs than with ruminants.

There was marked loss of appetite which developed soon after administration and lasted indefinitely in cases which proved fatal. In ruminants tympany occurred in several animals and signs of pain were apparent. A few hours after the animal was dosed, a state of lethargy developed which progressed to a deep coma, this lasting many hours in some cases. There were no convulsions, but the respiratory rate was found to rise by 25 per cent. within four hours of administration, and the majority of animals showed a rapid respiratory rate which sometimes degenerated into a wheezy distressed breathing when they were in a comatose condition. Temperature changes were very variable and unreliable, owing to the difficulty of ascertaining the true normal in healthy excitable sheep and pigs. There was some evidence of an initial temperature rise followed by a fall as coma supervened, and the pulse rate rose initially and fell markedly as coma developed.

Post-mortem evidence may be gathered under the following headings :—

Respiratory System

Marked congestion of the lungs in the majority of cases with interlobular oedema in a few cases. There was a pleural effusion of watery liquid in a minority of the animals examined, and blood splashes on the surface of the lung apparent in some cases.

Circulatory System

Congestion of all parenchymatous organs especially the liver, mammary gland and kidney in the majority of specimens. There was also a congestion of subcutaneous connective tissue in several cases, and petechiae along the coronary grooves in most of the animals. Petechiae were seen along the aortic wall in one specimen.

Stomach

In all cases the stomach contained the normal quantity of food. Gastritis was a marked lesion only in the pig. In ruminants small patches of inflammation could, in some specimens, be seen in the abomasum. (The acid-secreting compartment equivalent to the single stomach of the pig.)

Small Intestine

In the majority of cases the small intestine was empty of food, certainly those portions to which the phosphide had penetrated before death. Inflammation usually occurred and was intense, the distribution of inflammation possibly being associated with duration of life after dosing. In only one animal was the small intestine inflamed along more than half its length. Usually the inflammation had a discrete distribution of only a few feet (3 to 10), and this enteritis occurred in the duodenum or jejunum. Enteritis was characterised by congestion of the mucosa and haemorrhage into the bowel.

Large Intestine

No enteritis. Congestion in one animal.

Liver

In all cases the liver was swollen and extremely congested. In some the organ had a diffuse yellow, or putty colour ; in the others there was a patchy appearance with purple areas of congestion alternating with yellow areas of degeneration.

Excretory System

The kidneys were swollen and congested in the majority of animals, no other specific lesions being noted. The bladder mucosa in a few cases showed petechial haemorrhages.

Other Organs

The reproductive organs, mesentery, peritoneum showed no significant changes. The nervous system was not examined.

The lesions of major emphasis are (*a*) circulatory disturbance, congestion and breakdown being widely distributed over the body ; (*b*) liver damage showing acute fatty degeneration and congestion ; (*c*) lung changes showing congestion, and occasionally interlobular oedema ; (*d*) localised inflammation in the small, but not the large bowel. Points of interest are the lack of convulsions until the moment of death (anoxia), the unimportance of diarrhoea in contrast to many poisons, and evidence of respiratory embarrassment in some cases. The evidence clearly shows that a diagnosis of zinc phosphide poisoning without collateral chemical evidence is not advisable.

Summary

(1) Toxicity trials of red squill for cattle, sheep, pigs, goats, dogs and cats showed that goats were relatively the most resistant animals, but that, except for very young animals, it was unlikely that a toxic dose for normal livestock would be available after baits had been laid on a farm. Emesis can occur in some species, *e.g.*, pigs, dogs and cats, after eating the poison, but may not always save the animal.

(2) The symptoms of zinc phosphide poisoning were not specific, the general reactions being those of a

An Unusual Tumour

BY

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Subject

Manchester Terrier bitch, eight years old. Weight 30 lb. after operation.

History

The bitch was presented on October 14th, 1954, for examination with a history of enlargement of the abdomen which had become rapidly more pronounced during the last two weeks. She was in apparently normal health until the last few days when she lost her appetite and began to drink enormous quantities of water.

Clinical Signs

Upon examination the bitch appeared to be dull, with a normal temperature, a pulse rate of 100, and a slight pallor of the mucous membranes. Gross abdominal enlargement was obvious and was of a non-pendulous character. Palpation revealed a firm irregular mass which seemed to occupy most of the abdomen.

Differential Diagnosis

Pregnancy was considered as she had been in oestrum several weeks previously even though the owners said she had not been served ; but the nature of the enlargement was consistent neither with normal uterine contents nor with those of a gross pyometra.

Diagnosis

Abdominal tumour, probably of mesenteric origin, was put forward. Exploratory laparotomy was advised and undertaken 24 hours later.

Operation

Themalon, 250 mg. subcutaneously, induced deep sedation and anaesthesia was completed with ether inhalation.

The site was prepared in the usual manner and the abdomen opened in the mid-line by a 4-inch incision from the sternum towards the pubis. A very large tumour mass was immediately visible and it was necessary to enlarge the incision by 2 inches when it was possible to bring the tumour to the outside. It was then seen to consist of the spleen attached along its whole length to an enlarged mass, with a considerable amount of omentum involved.

Considering this enormous mass, more in faith than hope, we proceeded to ligate numerous vessels by which it was attached and severed them by blunt dissection.

Further exploration now revealed another tumour arising from the liver ; this appeared to have a pedicle and we were able to ligate and remove it together with a small portion of liver.

Excessive peritoneal fluid was removed, 200,000 units of intramammary penicillin introduced and the incision closed, using catgut internally and nylon for the skin.

The ether anaesthesia passed off rapidly ; 1 c.c. lethidrone antagonised the themalon and in a few minutes the bitch was able to walk around the surgery.

Recovery was uneventful and the skin sutures were removed seven days later. When last seen on November 29th, 1954, the bitch was still in good health.

The tumour weighed 4½ lb.

Pathological Report

Macroscopic Examination

The dorsal end of the spleen was contracted and measured 15 cm. long by 4 cm. wide by 1·5 cm. thick. On cut surface it was moderately cellular, the white pulp could be seen as pin-point white foci. Replacing the ventral extremity there was a large nodular tumour

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toxaemia. The evidence clearly showed that a diagnosis of zinc phosphide poisoning without collateral chemical evidence is not advisable.

Acknowledgments.—We are grateful to Dr. A. W. Stableforth and Dr. H. H. Green for their help. We are indebted to M. H. Moore and L. N. Ivins for technical assistance.

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(Part One of this article was published last week)

mass measuring 15 by 11 by 10 cm. The omentum was closely adherent to the capsule of this mass in many regions. A cut surface of the spleen and tumour showed that there was no distinct border between the splenic substance and the tumour tissue. The tumour was composed of tracts of oedematous fibrous tissue separating numerous cysts of various sizes ; some of these cysts contained serum while others contained blood coagula. Near the junction with the normal spleen the fibrous tissue strands were thinner and the tissue between them was white, soft and cellular, resembling lymphoid tissue.

The tumour arising from the liver measured 12 cm. long by 7 cm. wide by 4 cm. thick. On dissection it could be seen to be formed by a fold of tissue 2 cm. thick and of the consistency of adipose tissue. The colour of the cut surface of this tissue was white with areas of slightly pink firmer tissue.

The portion of liver showed a uniform pale yellowish colour, with distinct lobulation, and no focal lesions.

Histological Examination of Paraffin Wax Sections

The spleen is contracted with numerous megakaryocytes and macrophages containing haemosiderin distributed throughout the red pulp. The capsule of the spleen continues round the tumour mass and from it normal trabeculae run into the tumour tissue. The omentum adhering to the capsule appears normal. The cellular pattern of the tumour mimics that of normal spleen in showing large masses of lymphocytes surrounding arterioles, these masses being separated from one another by slightly engorged red pulp. Superimposed on this pattern there are tracts of oedematous fibrous tissue rich in distended lymphatic vessels and islands of plump spindle-shaped cells set in a pale blue matrix showing few collagen fibres. These regions resemble metaplasia of fibromatous element to form immature cartilagenous tissue. Centrally the tumour shows large areas of coagulative necrosis associated with thrombosis of blood vessels. There is no distinct demarcation between the tumour and the normal splenic tissue.

The tumour tissue arising from the liver is composed of adipose tissue showing fat necrosis and whorls of plump spindle-shaped cells distributed around blood vessels. This pattern is similar to that seen in some areas of the splenic tumour.

The liver pigment shows widespread centrilobular fatty change and early periportal fibrosis.

Comment

Small nodules of hyperplastic lymphoid tissue are frequently found in the splenic substance of elderly dogs. This tumour is possibly related to such hyperplastic nodules.

Acknowledgment.—We wish to accord our thanks to Mr. K. W. Head, Department of Veterinary Pathology, Royal (Dick) School of Veterinary Studies for his examination and report.

The Anthelmintic Efficiency of Piperazine Adipate* against *Neoascaris vitulorum* (Goeze, 1782)

A PRELIMINARY REPORT BY

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Introduction

NEOASCARIS VITULORUM is a helminth of cosmopolitan distribution and in tropical countries it is reported as a serious pathogen of young calves. There are few references to the economic importance of the parasite in recent literature but the position recorded earlier is unlikely to be greatly changed. For instance, Griffiths (1922) states that gross infestations are of common occurrence in East and South-Central Africa and that mortality rates are high in untreated cases. Hornby, quoted by Boulenger (1922), found the parasites very commonly in Northern Rhodesia and when present in large numbers they caused scouring, wasting and death. In Malaya, Lancaster (1954) considers *N. vitulorum* to be a very troublesome pathogen of cattle and water buffaloes and responsible for ill health and death losses. In an earlier communication Schwartz (1922) describes a similar situation in the Philippine Islands. At Coimbatore, in Madras, Vaidyanathan (1949) observed the condition as the most frequent disease of calves attending the College Veterinary Hospital.

In Nigeria the parasite is common, and within the past two years numerous cases in calves aged two weeks to five months from widely distributed areas have been diagnosed in this laboratory by faecal egg counts.

At Vom Stock Farm, where calves are reared on a high plane of nutrition, relatively heavy infestations are not associated with obvious clinical symptoms, but under "bush" conditions animals suffering from diarrhoea and unthriftiness have shown ascarid egg counts of 10,000 to 80,000 per gramme of faeces. Reports from field officers also indicate that the condition causes unthriftiness and severe death losses in certain areas, and infestations may be so heavy that masses of worms occlude the intestine.

According to Monnig (1947) no very satisfactory treatment is known. Hornby, quoted by Neveu-Lemaire (1936), recommends oil of turpentine on two successive days followed by a third dose one week later. The present writer, however, considers this treatment too complicated for use under field conditions.

Oil of chenopodium in castor oil has also been recommended and tests were carried out recently at Vom to assess the value of the drug administered as a single dose in castor oil. Results showed this treatment to be only partially effective. It is obvious, therefore, that a simple, more efficient treatment is required for Nigerian conditions and in view of the anthelmintic action of piperazine adipate against other ascarids of domestic animals (Sloan, 1954) it was decided to test the drug against *N. vitulorum.*

* Coopane—a product of Messrs. Cooper, McDougall & Robertson.

Methods

Piperazine adipate, prepared as a suspension of the drug in water, was administered as a drench, except in one toxicity test where yearling bovines were given the drug by subcutaneous injection. Experimental animals were not starved prior to drenching in toxicity and anthelmintic tests and in many cases dosing was carried out immediately after the morning feed.

In the initial toxicity test involving use of the drug at 0·1 to 0·4 gramme per lb. bodyweight the physical appearance of the faeces, feed consumption, eye reflexes, pulse and respiration rates were recorded twice daily for seven days after drenching.

In studying the effect of the drug at different dosage rates on faecal egg counts, zebu calves aged one to four months and weighing 100 to 220 lb. were used. They all harboured naturally acquired infestations of *N. vitulorum*. Faecal examinations were made on at least five consecutive days before dosing and were continued for 12 days after treatment. Faeces were removed manually from the rectum between 7 and 8 a.m. and the number of ascarid eggs per gramme was estimated by a modification of the McMaster method using a two-celled counting chamber. Two counts were made on each sample and the mean obtained.

During critical anthelmintic testing faeces passed by the experimental animals were collected every 24 hours for five days after treatment and all the expelled worms counted. The animals were slaughtered at the end of the fifth day and a thorough search was made for ascarids remaining in the alimentary canal.

Eggs for culturing were removed from the vagina and the first inch of the uterine tubes of worms passed in the faeces after dosing and incubated at 27° C. in Petri dishes containing a shallow layer of 2 per cent. formalin.

Results

(*a*) *Toxicity Tests*

Four zebu calves, four to six weeks old, and 47 to 85 lb. weight, which received the drug at 0·1, 0·2, 0·3 and 0·4 gramme per lb. bodyweight, showed soft faeces on the second, third and fourth days of the test but this was also common in control calves housed in the same building. The animal which received the drug at the highest concentration developed tympany and diarrhoea the day after treatment and on that day consumed only two-thirds of its normal milk feed. Diarrhoea persisted to the fourth day. This animal had shown slight tympany on a few occasions before the experiment began.

Following the initial test two groups, each consisting of 10 calves weighing 60 to 290 lb., showed no toxic reactions after receiving the drug at 0·1 and 0·2 gramme per lb. Piperazine adipate was later administered subcutaneously to two yearling bovines at the former dosage rate and apart from an extensive, oedematous swelling which developed at the site of inoculation and persisted for 36 hours no disturbance was observed.

(*b*) *Effect of Treatment on Faecal Egg Counts*

Test No. 1

Seven infected calves were available for the first test. Four received the drug at the rate of 0·2 gramme per lb. while three of similar ages and showing low, moderate and high egg counts respectively were left untreated to ensure that declining infestations were not involved.

The results are shown in Fig. 1 and it is seen that the mean egg counts of the treated calves showed an immediate, rapid reduction following dosing. All were negative for ascarid eggs by the sixth day and continued so for the remaining seven days of the test. Within 24 hours of treatment ascarid worms were passed

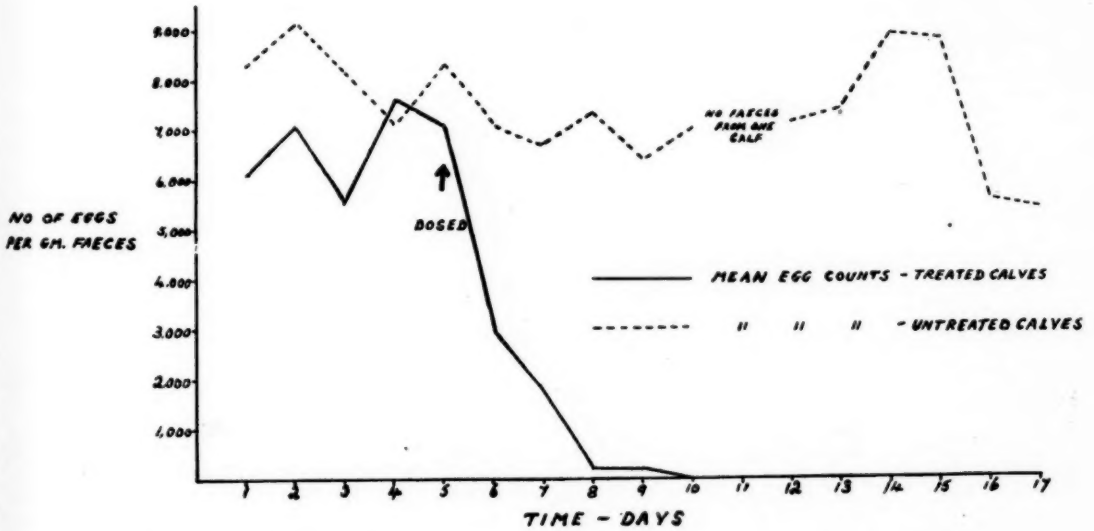

FIG. 1.—The effect of Piperazine adipate at 0·2 gm. per pound bodyweight on *Neoascaris vitulorum* egg counts.

in the faeces of these calves. The mean egg counts of the control animals were unaffected and no worms were found in their faeces.

Test No. 2

Seven calves were used in the second test and as weekly faecal examinations had been made on these animals from birth they were known to be harbouring stable ascarid infestations and it was therefore considered unnecessary to include controls.

Treatment with piperazine adipate at the rate of 0·1 gramme per lb. was followed by a spectacular decline in egg counts (Fig. 2). Three days after treatment only one calf was positive and by the fourth day all were negative and remained so until the test was terminated 12 days after dosing.

Again numerous worms were passed in the faeces.

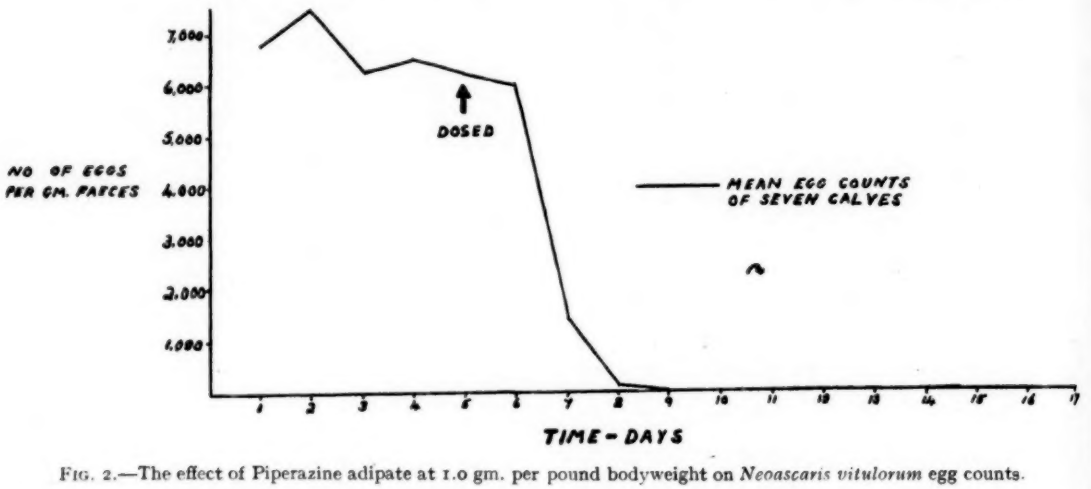

FIG. 2.—The effect of Piperazine adipate at 1.0 gm. per pound bodyweight on *Neoascaris vitulorum* egg counts.

(*c*) *Critical Anthelmintic Tests*

The results of critical anthelmintic tests at dosage rates of 0·1 gramme per lb. on mature and immature infestations are summarised in Table I.

TABLE I

CRITICAL ANTHELMINTIC TESTS WITH PIPERAZINE ADIPATE AGAINST *Neoascaris vitulorum* AT DOSAGE RATE OF 0·1 GRAMME PER LB. BODYWEIGHT

Days	Number of worms passed daily in faeces following dosing						Number of worms found at P.M.E. on 6th day	Per cent. efficiency
	1	2	3	4	5	Total		
Calf:								
3161	11	17	6	Nil	Nil	34	Nil	100
3202	22	3	Nil	Nil	Nil	25	Nil	100

In calf 3161, of 34 worms passed 20 were suitable for detailed examination and they were found to be mature specimens. Thirteen females measured 19 to 30 cm. and seven males 13 to 19 cm. long. The other 14 worms expelled were too disintegrated for detailed study.

Calf 3202 passed 25 ascarids between treatment and slaughter. Six females measured 7 to 17 cm. long and only two of these appeared likely to be egg-laying. Seven males measured 7 to 12 cm. in length. The remaining 12 specimens were disintegrating when passed but all were small, delicate worms. No ascarids were recovered *post mortem*.

The lengths of adult *N. vitulorum* given by Ransom (1911) are, females 22 to 30 cm. and males 15 to 25 cm. Measurements recorded by Boulenger (1922) working on specimens from the Punjab and from Northern Rhodesia are, females 21 to 27 cm. and males 17·5 to 27 cm. long. On the criterion of length alone it is therefore obvious that at least the majority of the ascarids expelled by calf 3202 were immature.

(*d*) *Effect of Treatment on Development of Eggs*

Eggs removed from nine worms passed in the faeces following dosing at both concentrations developed normally in artificial cultures and the embryonation rate was 90 to 95 per cent.

Discussion

The present work indicates that piperazine adipate in single doses administered to calves without previous starving is a safe anthelmintic and has an efficiency of 100 per cent. against *N. vitulorum*. The activity of the drug against immature worms enhances its value particularly where strategic systems of medication, based on a knowledge of the course of ascarid infestations, are required to reduce the quantity of infective material available to subsequent generations of calves.

The failure of the drug to inhibit development of eggs within the worms expelled requires that some action should be taken to sterilise the faeces passed during the four to five days after treatment. In view of Schwartz's finding (1922) that tropical sunlight is lethal to *N. vitulorum* eggs after one hour's exposure it is probable that if treated animals were run on open range for at least five days after dosing eggs voided as a result of treatment would be destroyed within the pre-fective stages of development.

Summary

1. Piperazine adipate administered to calves at rates of from 0·1 to 0·3 gramme per lb. bodyweight produced no toxic reactions.

2. When administered at the rate of 0·1 or 0·2 gramme per lb. to calves harbouring *N. vitulorum* infestations eggs disappeared from the faeces within five days of treatment.

3. Critical anthelmintic tests showed it to be 100 per cent. effective against immature and mature *N. vitulorum* at dosage rate of 0·1 gramme per lb.

4. The drug had no effect on the development of eggs removed from worms expelled as a result of these treatments.

Acknowledgments.—The author is indebted to the Cooper Technical Bureau for providing samples of the drug and to Mr. J. E. N. Sloan of that organisation who supplied information on the anthelmintic properties of piperazine adipate and suggested dosage rates. Acknowledgment is also due to Mr. J. Armour, Veterinary Officer, who carried out most of the clinical observations, to Mr. R. A. O. Shonekan who conducted the oil of chenopodium tests, and to the Director of Research for permission to publish this paper.

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BOVINE DENTISTRY

Klussendorf emphasises the importance of the periodic examination of teeth in cattle. In most animals there is a tendency for hooks to develop on the inner edge of the lower molars, and the outer edge of the upper molars. Although these hooks are usually of little significance, they can cause laceration of the tongue or cheek, and interfere with mastication. Another common dental abnormality, which may restrict jaw movement, is a persistent deciduous molar, which becomes fixed as a crown to the permanent molar.

KLUSSENDORF, R. C. (1954). *N. Amer. Vet.* **35.** (11). 827–828.

INDEX TO THE RECORD

May we make it known to our readers that the index to the 1954 volume of THE RECORD is in an advanced stage of preparation and should soon be printed. It is being compiled on new, and we believe better lines, and we hope will give satisfaction.

Slaughter of Casualty Animals

The National Farmers' Union, in the course of correspondence with the B.V.A. on the above matter, have recently stated that it would be greatly appreciated by them if veterinary surgeons, when advising producers to send animals for slaughter, would inform them that such animals, whenever practicable, should be sent to centres where the animals can be certified for the collective guarantee payments. Facilities for dealing with such animals have, they state, been considerably improved, and a list of present certification centres is given below.

Centres at which a Ministry of Food Grader is in attendance during normal office hours 9 a.m. to 5.30 p.m.

South-east Area

Arlesey, 41, High Street, Arlesey, Bedfordshire; 228. Brighton, Abattoir, Hollingdean Road, Brighton; 59385. Cambridge, Co-op. Abattoir, Sleaford Street, Cambridge; 3726. Canterbury, Broad Oak Road, Canterbury; 4074. Chelmsford, Co-op. Abattoir, Oval Lane, Chelmsford; 4148. Colchester, Co-op. Abattoir, Sheepen Road, Colchester; 2214, 2904. Croydon, Abattoir, 33, Cuthbert Road, Croydon; 2985. Guildford, Government Slaughterhouse, Woodlands Road, Guildford; 66765. High Wycombe, Easton Street Slaughterhouse, High Wycombe; Gt. Missenden 2226. Ipswich, Lady Lane, Ipswich; 51107. Islington, Metropolitan Cattle Market; North 5591. King's Lynn, Highgate, King's Lynn; 2942. Letchworth, Letchworth Bacon Co., Works Road, Letchworth; 818. Little Wratting, Sainsbury's Slaughterhouse, Little Wratting, Suffolk. Norwich, Red House, Aylsham Road, Norwich; 24967. Reading, Abattoir, Abattoir Road Reading; 60086. St. Neots, 10, Berkeley Street, Eynesbury, St. Neots. Waltham Abbey, Little Stewardstone Street Slaughterhouse, Waltham Abbey; Waltham Cross 2192. Watford Weddel & Co., 76, High Street, Watford; 9744. Woolwich, R.A.C.S. Abattoir, Garland Road, Woolwich; 2278.

South-west Area

Barnstaple, Barry & Sons, Tuly Street, Barnstaple; 2818. Bristol, Corporation Abattoir Gordon Road, Whitehall, Bristol; 56227. Exeter, City Abattoir, Tudor Street, Exeter; 54956. Fareham, Funtley, Nr. Fareham; 2110. Plymouth, City Abattoir, Maxwell Road, Prince Rock, Plymouth; 2723. Redruth, c/o Livestock Ltd., Drump Road, Redruth; 781. Salisbury, Abattoir, Churchfields Road, Salisbury; 5938. Swindon, Abattoir, Melborough Lane, Swindon; 5403. Taunton, 28, High Street, Taunton; 2723. Wimborne, Udden's Cross, Nr. Wimborne; Ferndown 856.

Wales Area

Aberdare, Public Slaughterhouse, Ynys Road, Aberdare; 497. Abergavenny, Co-op. Slaughterhouse, Market Yard Abergavenny; 21. Brecon, Public Slaughterhouse, Canal Bank, Brecon; 319. Brynmawr, 50, Bailey Street, Brynmawr; 251. Caernarvon, Municipal Abattoir, Balaclava Road Caernarvon; 148. Cardiff, Public Slaughterhouse, Roath Market, Cardiff; 25115. Carmarthen, Public Abattoir, Fair Lane, Carmarthen; 7251. Llanelly, Public Abattoir, Swansea Road, Llanelly; 487. Llandudno, Municipal Abattoir, Builder Street, Llandudno; 7200. Colwyn Bay, Municipal Abattoir, Mochdre, Colwyn Bay; Colwyn Bay 44740. Neath, Slaughterhouse Eastland Road, Neath; 728. Rhyl, Municipal Abattoir, Ffordd Las, Rhyl; 628. Newport (Mon.), Public Abattoir, Wyndham, Newport; 5782. Swansea, Public Abattoir, Dyfatty Street, Swansea; 55410. Ton Pentre, Govt. Slaughterhouse, Llanfoist, Ton Pentre; 2123. Wrexham, Municipal Abattoir, Wrexham; 3340.

Midland Area

Birmingham, City Meat Market, Bradford Street, Birmingham; Midland 2834. Boston, 28, Main Ridge, Boston. Chesterfield, Municipal Abattoir, Cattle Market, Chesterfield. Derby, Municipal Abattoir, Cattle Market, Derby; 46041. Grimsby, Govt. Abattoir, Cromwell Road, Grimsby; 56501. Hereford, Corporation Abattoir, Hereford; 3546. Leicester, C.W.S. Abattoir, Aylestone Road, Leicester; 58410. North-

ampton, 8, Regent Square, Northampton; 4693. Nottingham, Corporation Abattoir, Cattle Market, Nottingham. Scunthorpe, Municipal Abattoir, Cattle Market, Scunthorpe. Shrewsbury, Corporation Abattoir, Roushill; 3090. Stoke-on-Trent, Municipal Abattoir, Bethesda Street, Henley, Staffs; S.O.T. 25171. Stratford-on-Avon, 6, Chapel Street, Stratford-on-Avon.

North-west Area

Barrow, Corporation Abattoir, Cavendish Street, Barrow; 636. Birkenhead, Woodside Lairage, Birkenhead; 6680. Blackpool, Corporation Abattoir, Abattoir Road, Blackpool; 28338. Leigh, Bucks Street, Leigh; 820. Liverpool, Stanley Abattoir, Prestcot Road, Liverpool, 13; Stonycroft 4225. Manchester, City Abattoir, Water Street, Manchester; Blackfriars 7004. Preston, Corporation Abattoir, Brook Street, Preston; 7488. Tranmere, Tranmere Abattoir, New Chester Road, Tranmere; Birkenhead 1811.

North-east Area

Barnsley, Bankers Hill, Pontefract Road, Barnsley; 2774. Bedale, Wensleydale Wholesale Meat Co., Aiskew, Bedale; 165. Berwick-on-Tweed, Municipal Slaughterhouse. Tweed Street, Berwick-on-Tweed. Bradford, 33, St. James's Abattoir, Bradford, 1. Darlington, Corporation Abattoir, Park Lane, Darlington; 4143. Doncaster, Corporation Abattoir, Market Place; 49097. Hexham. Council Slaughterhouse, Hexham; 673. Hull, C.W.S. Slaughterhouse, Gt. Thornton Street, Hull; 16865. Leeds, Wholesale Meat Market, New York Street, Leeds; 23034. Newcastle-upon-Tyne, L. A. Slaughterhouse, Cattle Market (off Scotswood Road); 27550 Ext. 63. Rotherham, Corporation Abattoir, Corporation Street, Rotherham; 2499. Sheffield, Corporation Abattoir, Sheffield; 20939. West Hartlepool, Barnard Street, West Hartlepool; Hartlepool 3798. York, Cemetery Road, York; 3654.

Scotland

Aberdeen, Slaughterhouse, Deer Road, Woodside; 20457. Arbroath, Municipal Slaughterhouse, Arbroath. Biggar, Public Slaughterhouse, Station Road, Biggar; 229. Buckie, Municipal Slaughterhouse, Buckie. Dumfries, Public Slaughterhouse, Huntingdon Road, Dumfries; 1541. Dundee, Slaughterhouse, Dock Street, Dundee; 81315. Edinburgh, Public Slaughterhouse, Gorgie, Edinburgh; Craig Lockhart 3991. Elgin. Municipal Slaughterhouse, Elgin. Fraserburgh, Slaughterhouse, Fraserburgh; 557. Glasgow, 50, Melbourne Street, Glasgow; 1296. Greenock, Public Slaughterhouse, Greenock; 200591. Invergordon Slaughterhouse, Invergordon; 131. Inverness, Slaughterhouse, Inverness. Kelso. Public Slaughterhouse, Spylaw Road, Kelso; 262. Kirkaldy, Slaughterhouse, Kirkaldy; 2827. Motherwell, Public Slaughterhouse, Motherwell; 563. Perth, Municipal Slaughterhouse Shore Road, Perth. Stirling, McGrouther Ltd., Cornton Road, Stirling; 708/9.

This list will probably be amended from time to time.

RINGWORM

In last week's issue of THE RECORD a note regarding ringworm was published, and it was mentioned towards the end that Weybridge would be providing facilities for a diagnostic service in both large and small animals and that material should be sent there clearly labelled.

Arrangements are in fact being made for setting up a veterinary mycological reference laboratory at Weybridge, but it will be about two months before a diagnostic service in animal mycoses can be offered to members of the profession and other interested workers.

It is hoped that members will be interested to send samples in due course, but *please note that these will not be acceptable until about the end of April.*

POLICEMEN ON HORSEBACK

(*Reprinted by courtesy of "The Times"*)

IN these days of mechanisation there is some justification for asking why 24 police forces in England should each continue to maintain a mounted branch. Is the mounted policeman more than a picturesque survival from past days? The simplest answer is that mounted police are unrivalled in the work of crowd control; it is commonly estimated that for this purpose one mounted policeman is worth 12 to 15 foot policemen.

Why then, it may be asked, does not every county or borough police force of any size have a mounted branch? In fact, only 22 of the 111 county and borough forces, in addition to the Metropolitan Police and the City of London police, have a mounted branch. They are concentrated almost wholly in the industrial cities and "conurbations" of the north and the midlands. South of Birmingham the only mounted branch outside Greater London is in the city of Bristol, with 11 horses, except that Southend has two horses and Eastbourne one.

Two Groups

The mounted police of England can be divided into two groups almost equal in numbers: the Metropolitan Police mounted branch with 199 horses, and the 23 other forces with 216 horses. The City of London force has only seven horses, and both men and horses train with the Metropolitan at Imber Court. Apart from the minor contributions of Southend and Eastbourne and the larger mounted branch at Bristol, the picture moves far away from the Metropolitan district. The Lancashire county force has 26 horses; the city force of Liverpool has 24, Manchester 20, Bolton six, and Salford four. The West Riding force has 23 horses; Leeds city force has 12, Bradford and Sheffield seven each, and Hull four. Durham county force has 12 horses and Newcastle upon Tyne six. Birmingham has 24 horses, Staffordshire six, and Stoke four. Nottingham (4), Derby (3), Norwich (2), and Great Yarmouth (1) complete the total.

The existence of these mounted branches in such diversity in the county and borough forces shows that local authorities still play the leading part in their own policing. Nor is there any dull uniformity of pattern in administration. In Manchester, for example, the city's 20 horses are mounted by 12 policemen, but in Birmingham the city's 24 horses are mounted by 27 policemen. In other words, the deficiency of 268 men in the authorised strength of 1,459 in the Manchester police force is reflected in the manning of the mounted branch, while in Birmingham the deficiency of 320 men in its authorised strength of 2,060 is not allowed to make any difference to the mounted branch.

In The Metropolis

The Metropolitan mounted branch is also kept up to full strength in spite of the force as a whole being some 3,800 short of its authorised establishment of 19,667. It stands alone, however, in the scope of its duties both in crowd control and ceremonial participation in state occasions. The Commissioner of Police of the Metropolis is, on such occasions, a mounted officer, as also are the Assistant Commissioner and certain other officers.

In the county and borough forces outside London few, if any, chief constables ever mount a horse ; nor even does the officer in charge of the mounted branch.

In these forces, moreover, crowd control is mostly limited to such regular events as football matches, race meetings, and annual processions. Mounted police work is seen at its simplest in Bolton, where the borough force hires the six horses of its mounted branch from a privately owned riding school. It is an economical arrangement, costing the borough about £100 a year—that is, £50 as retaining fee for the six horses and £1 each time a horse is hired. They are hired two at a time for each of Bolton Wanderers' 24 home matches and for the civic procession each "Mayor's Day." Bolton's mounted police (two sergeants and five constables) are "on the beat" as foot policemen until detailed for one of the football matches, but the entire branch spends an annual period of five consecutive days at the riding school.

The cost of a full-time mounted branch varies from force to force but the figures for Manchester are no doubt a fair sample. There the city's 20 police horses cost about £2,500 a year for provender, bedding, farriery, stable utensils and the services of veterinary surgeons. The cost works out at much the same rate in Eastbourne, where the borough force maintain a single horse for patrolling the downs near Beachy Head. In the larger mounted branches £200 is usually reckoned the maximum price for a horse ; for £400 a police buyer would expect two exceptionally good horses or three of ordinary quality.

There is much rivalry in the buying of police horses. The Metropolitan force buys mainly in Yorkshire, but in Leeds it is generally considered that the Yorkshire horses are too light and so Leeds tends to look to Ireland for "heavy hunters." Liverpool also buys mainly from Ireland but Manchester looks mostly "towards Berkshire." Birmingham also looks towards Berkshire, though it used to favour Irish horses, and also buys a good deal locally ; 17 out of the city's 24 horses are greys, and the city has an eye to the grey wherever it is to be bought. Lancashire county constabulary will buy a horse on its merits anywhere. Bolton, where the mounted branch is only indirectly concerned in buying, enjoys one police horse that was bought from a circus.

The Metropolitan mounted branch maintains patrols from 28 stables in the four districts of its vast area ; only 27 or so of its horses are kept at Imber Court for training and other purposes. The Lancashire mounted branch similarly has patrols in seven of the county's urban divisions, keeping seven of its horses at Hutton, the county headquarters near Preston, for training. In the West Riding also the mounted policeman is mainly a unit of urban policing. Birmingham, Liverpool, Manchester, and Leeds vary slightly in the extent to which they patrol the centre of the city, but in each case a point is made of sending patrols to new estates on the fringes of the city where people see much less of the police.

The Daily Patrol

It is a fair estimate that each of the five county or borough mounted branches with 20 or more horses deals with about 100 offences or "incidents" a year during patrol duty. This is an important claim when it comes to justifying the existence of mounted police forces. The daily mounted patrol gives man and horse about three and a half hours' exercise, which is naturally a main part of its purpose. But since the man on the horse is first and foremost a policeman on duty, the patrol also has its part in the prevention of crime.

The mounted branch of a police force is to-day recruited only from men who have done at least two years' probation on the beat. Few of them have had any previous experience with horses. About half the men in the mounted branches outside the Metropolitan district are from former cavalry or horse artillery regiments, but new recruits from these sources form a mere fraction of present entries.

There have been no major changes in the composition of the Metropolitan mounted branch since about 1931. Outside London the slight changes brought about by watch committees and county authorities may reflect local public opinion. Birmingham has increased its horses by three since 1952, but Lancashire has not replaced four horses given up in 1952. Hull revived its mounted branch in 1949. At least two forces since the war have incurred the displeasure of Her Majesty's Inspectors of Constabulary by parading small mounted sections that were mere show pieces with hired horses, and one chief constable made a mistake in asking that the mounted section be inspected first—which compelled him to reveal that the horses were urgently needed later for a funeral.

Book Review

Index of Treatment. W. HAMILTON KIRK. Third Edition 1954. Ballière, Tindall & Cox., London. Pp. 888. Figs. 194. Price 45s.

This well-known text-book now appears in its third edition and this continued demand is evidence that it meets the needs of the busy small-animal practitioner. The arrangement of the book follows the pattern of previous editions, in which Part I contains a number of chapters on various forms of therapy, Part II is an Index of Therapeutics in which many clinical conditions are listed alphabetically and their treatment discussed, and Part III has a number of miscellaneous chapters on practical matters such as Handling and Restraint, Nursing the Sick, and Euthanasia.

New material includes a chapter on the Antibiotics, an extension of the section on the treatment of virus diseases and new analgesics and anaesthetics. Parts II and III, which are based on the author's extensive experience in small-animal practice, contain the knowledge which has made this book popular with the practising veterinary surgeon. Part I, which is of a more theoretical nature, is not so valuable. Some of the information is out of date and does not contribute to the value of the work. The book now contains a list of proprietary preparations and their makers ; if this list contained the active principles of these preparations it would supply useful information which would also allow the author to drop the irritating use of proprietary names in the main text and still enable readers to ascertain useful sources of supply.

Despite these drawbacks the book will continue to supply the need of those who want readily available information based on practical experience.

The R.C.V.S. Council Election

The election will shortly take place of five members of Council, R.C.V.S. The following gentlemen have offered themselves as candidates :

Mr. G. Atkinson, of Exeter, Professor T. J. Bosworth, of London, Mr. J. A. Dall, of Torquay, Professor H. G. Lamont, of Belfast, Mr. J. B. White, of Salisbury, Mr. H. M. Wilson, of Cupar-Fife, and Dr. W. R. Wooldridge, of Stanmore.

It has been thought useful to revive the previous practice of publishing brief biographical details of candidates for election to the Council of the Royal College, and these are as follows :

Mr. G. ATKINSON, M.R.C.V.S., was born in South Shields in County Durham and was educated in Edinburgh and at the Royal Veterinary College, London, qualifying from the latter in July, 1914. After a few months in general practice he joined the Northumbrian Territorial Division as Veterinary Officer and went with them to France in the early part of 1915. He served in France, Mesopotamia and India (North-West Frontier).

Demobilised in November, 1919, he then joined the Ministry of Agriculture as Veterinary Inspector in February, 1920. Before being appointed to Exeter as Superintending Veterinary Officer in 1938, the principal places in which he was stationed were the Laboratory at Weybridge (four years), Bristol (four years), Head Office, London (four years) and Newcastle-on-Tyne (three years). Mr. Atkinson was promoted to Divisional Inspector in 1925 while at the Laboratory, and to Superintending Veterinary Officer in 1938. He joined the B.V.A. in the early nineteen-twenties and has been a member of the Council for about five years. He serves on the Veterinary State Medicine Committee. Mr. Atkinson is a member of the Mid-West and Western Counties Divisions of the B.V.A. and has been Chairman of the latter. He is a member of the Rotary Club of Exeter and an associate member of the Royal Sanitary Institute.

Professor T. J. BOSWORTH, M.A., B.SC., M.R.C.V.S., D.V.S.M., was born on March 21st, 1898. He was educated at Bedford Modern School and the Royal Veterinary College, graduating in 1915. He served in the R.A.V.C. from 1917 to 1919 ; was Research Assistant, Royal Veterinary College, 1920–1923 ; Head of Veterinary Dept., Wellcome Physiological Research Laboratories, 1923 ; Assistant Director, Institute of Animal Pathology, Cambridge, 1924–1939 ; Professor of Pathology and Director of Research Institute in Animal Pathology, Royal Veterinary College, 1931–1951. He is Joint Editor of the Journal of Comparative Pathology and Therapeutics ; Member of Council, Imperial Cancer Research Fund ; Founder and Past-President of the Veterinary Research Club ; Past-President, Royal Counties V.M.A., Association of Veterinary Teachers and Research Workers (Southern Division) and Section of Comparative Medicine, Royal Society of Medicine. He was sometime examiner to the Royal College of Veterinary Surgeons and to the Universities of Cambridge, Edinburgh, Liverpool, London, and the National University of Ireland. Professor Bosworth was a member of B.V.A. Council, from 1944–1954, and has been a member of the R.C.V.S. Council, since 1944.

Mr. J. A. DALL, B.SC., M.R.C.V.S., was educated at Daniel Stewart's College, Edinburgh, and the Royal (Dick) Veterinary College. He qualified in 1937 and was awarded his B.Sc. (Edinburgh) in the following year. In 1940 he became a partner in Masson & Dall, Veterinary Surgeons, of Torquay. He was elected a member of the Council of the Western Counties Veterinary Association, of which he became President in 1950. On the death of the late S. J. Motton Mr. Dall undertook the Secretaryship of that Association, and in this capacity he was Local Secretary to the B.V.A. Congress at Torquay last year. He is a member of the Council of the B.V.A. and serves on the Organising Committee.

Professor H. G. LAMONT, O.B.E., D.SC., M.R.C.V.S., was born in 1901. He qualified at Queen's University in 1923 and was awarded his B.Sc. in 1926, M.Sc. in 1928 and D.Sc. in 1940. In 1950 Professor Lamont received the O.B.E. for services to agriculture and veterinary science in Northern Ireland.

Professor Lamont was Demonstrator in the Department of Pathology of the Medical Faculty, Queen's University, from 1924–1926, and joined the Veterinary Research Division in that year. He became Director in 1931, being in charge also of the Dairy Bacteriology Department 1930–1934. First lecturer, and later Reader in veterinary science he became Professor in Comparative Pathology at Queen's University in 1951.

During that year Professor Lamont was elected to the Council of the Royal College, becoming Chairman of the Examination and Education Committee in 1952. He had been a member of the Board of Examiners since 1945. He was President of the North of Ireland Veterinary Association in 1941 and again in 1955, and was President of the North of Ireland Tuberculosis Association in 1952, 1953 and 1954.

Professor Lamont is the son of a veterinary surgeon and is keenly interested in veterinary practice. He has published many papers on veterinary subjects, particularly on the diseases of pigs.

Mr. J. B. WHITE, M.R.C.V.S., was born in Aberdeen in 1906, the son and grandson of veterinary surgeons. He came south to Petersfield at an early age and was educated at Churcher's College. He entered the Royal Veterinary College in October, 1923, and qualified in July, 1927, being amongst the last students to study under Sir John M'Fadyean. After a short spell as locum tenens he was assistant to E. R. Smythe, of Falmouth, and in March, 1930, became joint assistant to Messrs. E. R. Harding and H. C. Rockett, of Salisbury. The two practices were amalgamated and Mr. White eventually became senior partner.

Always interested in his local Division, he was for several years Secretary, and then President of the Southern Counties Division. He has served latterly

as Council representative on the B.V.A. of whose Technical Development Committee he is a member. Mr. White was elected to serve on the Council of the Royal College two years ago.

Mr. HARRY M. WILSON, M.R.C.V.S., is a native of Edinburgh, where he was educated at George Watson's College for boys and at the Royal (Dick) Veterinary College. The whole of his professional life has been spent in general practice, mainly large animal.

He is a member of Council of the Scottish Metropolitan Division of the Scottish Branch of the B.V.A. and is one of the Division's elected members of Council of the B.V.A. He is also a member of the General Purposes and Finance Committee and of the Veterinary State Medicine Committee. He has served on the Council of the Scottish Branch of the B.V.A. and is the Branch's representative on the Advisory Committee on Artificial Insemination, Department of Agriculture for Scotland. He is a member of Council of the National Veterinary Benevolent and Mutual Defence Society.

Mr. Wilson is a member of the Department of Agriculture for Scotland panel of Veterinary Referees in respect of the licensing of bulls, and is one of the Department's Veterinary Inspectors in respect of the licensing of stallions.

Dr. W. R. WOOLDRIDGE, this year's President of the Royal College, was born in 1900. He was educated at Sir Walter St. John's School, the Royal Veterinary College, Birkbeck College, London, and Caius College, Cambridge. Having taken his B.Sc. in chemistry, he then studied for the professional diploma, which he took in 1924. He was awarded his M.Sc. (1927), followed by the Ph.D. (Cantab.) in 1928. In the following year he was appointed to the London School of Hygiene and Tropical Medicine, first as Demonstrator and soon afterwards as Lecturer in the Division of Biochemistry. In 1931 he became President of the Veterinary Research Club and in the same year was elected to the Council of the B.V.A. He has remained on the Council ever since, serves at present as Chairman of its Parliamentary and Public Relations Committee, and is also a member of the Technical Development Committee. He is a Governor of Birbeck College, a Governor of the London School of Hygiene and a Governor of the Royal Veterinary College. In May, 1952, he was elected to the University of London Senate. In 1942 Dr. Wooldridge founded the Veterinary Educational Trust, which later became the Animal Health Trust, of which he is the Scientific Director.

Abstracts

A Barbiturate Antagonist. HARRIS, T. A. B. (1955). *Lancet*, January 22nd, 1955, 181.

This paper opens with a reference to the discovery by Shaw of a barbiturate antagonist, $\beta\beta$-methyl-ethyl glutarimide, which has been designated NP 13.

In rats, mice, and rabbits, NP 13 at a dosage rate of 15 mg. per kg. bodyweight antagonised pentobarbitone, thiopentone, and barbitone anaesthesia reducing the sleeping time by half and doubling the barbiturate-depressed respiration rate. In unanaesthetised animals, NP 13 produced fasciculations and generalised convulsions. In these animals NP 13, at a dosage rate of 30 mg. per kg. bodyweight, caused fatal convulsions, which could be prevented or abolished by pentobarbitone anaesthesia. Shaw reported that NP 13 significantly shortened the period of coma in 39 human cases of barbiturate poisoning.

The present paper reports the successful use of NP 13 in 73 human cases. In 57 cases NP 13 was used to shorten the period of thiopentone anaesthesia in patients subjected to electrical convulsive therapy. In six cases NP 13 was used to shorten the period of thiopentone anaesthesia following orthopaedic manipulations. In 10 surgical cases, NP 13 was used following anaesthesia with thiopentone and cyclopropane. In these cases, there was a delay in response that can be attributed to the depressant action of cyclopropane anaesthesia and surgical shock.

The author concludes that NP 13 is quite innocuous using doses of 50 mg. for the human subject and claims that NP 13 is a barbiturate antagonist of real clinical worth. G. F. B.

Treatment of Lead Poisoning with Calcium Disodium Versenate. GILES, H. MCC., MOORE, C. J., & STILL, BESSIE, M. (1955). *Lancet*, January 22nd, 1955. 183-185.

The use of the calcium complex of ethylenediamine tetra-acetic acid (calcium disodium versenate) in the treatment of lead poisoning has been reported by a number of authors in the U.S.A. since 1952. The present report deals with the successful treatment of lead poisoning in a child of two and a half years. Calcium disodium versenate was given intravenously at 12 hourly intervals in doses of 0.5 g. in 200 ml. of 0.18 per cent. sodium chloride in 4.3 per cent. dextrose, each intravenous infusion lasting some two hours.

Calcium disodium versenate is the calcium complex of ethylenediamine tetra-acetic acid (E.D.T.A.). E.D.T.A. is a chelating agent, *i.e.* a substance capable of binding multivalent metallic ions so as to cause their functional removal from solution. The stability of complexes so formed varies with the metal concerned, and a metal forming a stronger complex can replace one forming a weaker complex. Thus, lead displaces calcium from the calcium sodium complex, which for this reason is used in the treatment of lead poisoning. The calcium complex must be used because E.D.T.A. alone would combine with calcium in the plasma so rapidly as to cause hypocalcaemic tetany and death. Experiments on animals have shown that calcium disodium versenate causes no serious reactions.

The dosage is at present rather empirical but it is suggested that the drug should be given intravenously not faster than 0.5 g. per 30 lb. bodyweight per hour.

The daily dose should not exceed 1 g. per 30 lb. bodyweight and the weekly dose should not exceed 5 g. per 30 lb. bodyweight. Courses should be limited to 7.5 g. per 30 lb. bodyweight, given over 10 days, with seven-day rest periods between the courses. The drug should not be injected in concentrations of more than 3 per cent. It is computed that 1 g. of calcium disodium versenate will bind 553 mg. of lead. E.D.T.A. is excreted by the kidneys and, in rats, 99 per cent. of an intravenous dose is recoverable in the urine.

Administration of ammonium chloride during versenate treatment did not increase the urinary excretion of lead. G. F. B.

(Calcium disodium versenate is supplied by Riker Laboratories, Loughborough.)

News and Comment

GLASGOW UNIVERSITY V.M.A. ANNUAL BALL

The sixth annual ball of the Glasgow University Veterinary Medical Association was held in the Rhul Restaurant, Glasgow, on February 11th. Among the guests were Professor Robertson, President of the B.V.A. and Mrs. Robertson; Professor and Mrs. Weipers; Inspector Brock, of the Glasgow Mounted Police; and Mr. Roger Mason and partner from the Bristol School.

There was a record attendance both of staff and students this year, and the strains of a hunting horn announced that a number of graduates, many of whom had been circularised, had joined the gathering. Prizes were presented by Mrs. Weipers, who, in turn, received a bouquet of red carnations from the President's lady.

The evening was a great success, and a happy augury for the Ball in 1956.

UFAW ACTIVITIES

The Federation's Easter Congress will be held at St. Patrick's Hall in the University of Reading, from April 1st to 4th. Among matters which will be of interest to members of the profession will be a visit to the Agricultural Research Council's Field Station, a paper on The Care and Management of Bulls, by Dr. N. S. Barron, a Historical Retrospect of Electrocution, by Major C. W. Hume, and a paper on some recent developments with regard to Animals and the Law, by T. G. Field-Fisher, Barrister-at-Law. The inclusive fee for the Congress, which includes full board and the expenses of various expeditions, is three guineas, which is reduced to £1 10s. for students. Applications for attendance and all enquiries should be addressed to :—

Miss M. P. Williams, UFAW, 7a, Lamb's Conduit Passage, London, W.C.1 (CHAncery 9221).

A symposium on the Federation's scientific work for animals was held at University College, Gower Street, on the evening of February 11th. The papers given included those by Major Hume on The Humanitarian Aspect of Rabbit Control; by Dr. Jean Vinter on The Evolution of a Humane Method of Killing Unwanted Cats and Dogs; and by Dr. W. M. S. Russell on A Research into the History and Progress of Humane Experimental Techniques. The Chairman for the symposium was Mr. Kenneth Bird, C.B.E. and it was followed by a sound film on the training of dogs.

ACCESSIONS TO THE R.C.V.S. LIBRARY

FESTSCHRIFT zur Feier des siebzigsten Geburtstages von Professor Dr. Walter Frei von seinen Freunden, Kollegen und Schuleren Gewidmet. Zurich, 1952.

INDIAN COUNCIL OF AGRICULTURAL RESEARCH. The I.C.A.R. silver jubilee souvenir, 1929–1954, December, 1954.

MAURITIUS, Colony. Annual report of the department of agriculture for the year 1953.

NASR, H. The nutritional significance of microbial synthesis in the pig caecum. Cairo, 1952.

NEW ZEALAND. Department of agriculture. Animal research division. Annual report 1953–54.

ORISSA. Annual report of the animal husbandry and veterinary department, Orissa, for the year 1951–52.

ST. KITTS–NEVIS–ANGUILLA. Annual report of the agricultural department for the year 1953.

UNITED NATIONS. World Health Organisation. Rabies [a number of articles on aspects of rabies, being W.H.O. Bulletin, vol. 10, no. 5] 1954.

UFAW. Twenty-eighth annual report, 1953–54.

MINISTRY OF AGRICULTURE VETERINARY FIELD STAFF

The following members have been appointed to the permanent staff as Veterinary Officers:—

Mr. G. M. Gates, M.R.C.V.S., stationed at Guildford; Mr. I. McLachlan, M.R.C.V.S., D.V.S.M., stationed at Chester; Mr. E. W. Russell, M.R.C.V.S., temporarily stationed at Gloucester.

FOOT-AND-MOUTH DISEASE

The area described below will cease to be an Infected Area for the purpose of preventing the spreading of foot-and-mouth disease on February 22nd, on which day the Foot-and-Mouth Disease (Infected Areas) Special Order No. 1, 1955, will be revoked.

The County Borough of Tynemouth.

County of Northumberland—The Boroughs of Blyth, Wallsend, and Whitley Bay, and the Petty Sessional Divisions of Bedlingtonshire and East Castle Ward.

Petty Sessional Division of West Castle Ward—The parishes of Stannington, Horton Grange, Brenkley, Mason, Dinnington, West Brunton, East Brunton, North Gosforth, Gosforth, and Longbenton.

MYXOMATOSIS IN HARES

It has been confirmed by the Veterinary Laboratory, Weybridge, that the carcase of a hare found in Northern Ireland was affected with myxomatosis. This is the first occasion on which myxomatosis has been confirmed in a hare in the United Kingdom. The hare in question is a mountain hare, also known as the blue or varying hare.

There is a fairly close relationship between hares and the European rabbit and because a few hares have been affected in France and Germany a close look-out has been kept for infection among hares in the United Kingdom. In view of the wide dissemination of myxomatosis among rabbits, both here and on the Continent, it is noteworthy that so small a number of cases has been confirmed in hares. No particular significance is to be attached to this new case. It was already known that the hare could contract myxomatosis. It is emphasised that there is no evidence that the disease affects other animals.

THE B.R.X. CLUB

The Annual Dinner of the B.R.X. Club is to be held at the Royal Veterinary College on Saturday, April 2nd. Professor G. H. Wooldridge will be the guest of honour, and the President, Professor C. W. Ottaway, will be in the Chair. Members should receive their formal invitations with reply slips by the end of February. Anyone who has been missed out and who wishes to attend the dinner should write direct to the Secretary—Mr. A. S. King, Hon. Sec., B.R.X. Club, Department of Veterinary Anatomy, The University, Bristol 8.

PERSONAL

Births

HARE.—On January 5th, 1955, to Audrey, wife of Dr. W. C. D. Hare, B.SC., PH.D., M.R.C.V.S., of Gullane, East Lothian, a son.

KEAY.—On February 9th, 1955, at Rotorua, New Zealand, to Margaret, wife of Anthony J. Keay, M.R.C.V.S., a daughter.

WRIGHT.—On January 30th, 1955, to Brenda, wife of G. W. Wright, M.R.C.V.S., at Sackville Memorial Hospital, Sackville, New Brunswick, a son, Anthony.

R.C.V.S. OBITUARY

BLACKWELL.—Walter Glynn, of 1, Exe View, Exmouth, Devon. Graduated London, December 21st, 1906. Died February 9th, 1955, aged 71 years.

LOMAS.—Harry, of Willow Cottage, Stanion, nr. Kettering. Graduated Edinburgh, May 21st, 1897. Died February 9th, 1955, aged 80.

STEVENSON.—John, of 60, Salisbury Road, Great Yarmouth, Norfolk. Graduated Glasgow, May 27th, 1899. Died February 5th, 1955, aged 79.

COMING EVENTS

23rd (Wed.). Joint Meeting of the Society of Practising Veterinary Surgeons and the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 2.30 p.m.

Annual General Meeting of the Scottish Metropolitan Division, B.V.A., in the Royal (Dick) School of Veterinary Studies, Edinburgh, 2.30 p.m.

24th (Thurs.). Meeting of the Essex Division, B.V.A., at the Essex Institute of Agriculture, Writtle, 7.30 p.m.

Spring Meeting of the Dumfries and Galloway Division, B.V.A., in the Station Hotel, Dumfries, 2.30 p.m.

25th (Fri.). Annual General Meeting of the Midland Counties Division, B.V.A., at the Birmingham Medical Institute, 154, Gt. Charles Street, Birmingham, 11 a.m.

Liverpool University Veterinary Society Annual Ball, in the Students' Union, Brownlow Hill, Liverpool 7, 8 p.m.

March

2nd (Wed.). Annual General Meeting of the Lancashire Division, B.V.A., in the Veterinary Hospital, University of Liverpool, 2.30 p.m.

Ordinary General Meeting of the Society for the Study of Animal Breeding, at the Wellcome Research Institute, 183-193 Euston Road, London, N.W.1, 11.30 a.m.

4th (Fri.). Annual General Meeting of the Royal Counties Division, B.V.A., in the New Letters Lecture Theatre, Reading University, 2.30 p.m.

9th (Wed.). Ordinary Meeting of the A.V.T. and R.W.'s (Northern Region) in the Veterinary Hospital, Pembroke Gardens, Liverpool 3, 2 p.m.

April

30th (Sat.). Association of State Veterinary Officers Annual General Meeting and Dinner in London.

May

6th (Fri.). Royal Veterinary College 163rd Annual Ball at the Hyde Park Hotel.

June

10th (Fri.). Annual Dinner of the R.A.V.C. Officers' Club, at Grosvenor House, Park Lane, London.

September

4th to 10th, 73rd Annual General Meeting and Congress of the British Veterinary Association at Belfast.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ayr. No. 2, South Craig, Kilmarnock (Feb. 7).

Cambs. Springfield, Barton Road, Wisbech (Feb. 10).

Ches. Swanley Cottage, Burland, Nantwich (Feb. 10).

Cumberland. Easley Home Farm, Brampton; Hudscales, Hesket Newmarket, Wigton (Feb. 8).
Durham. South Farm, Cornsay; Shield Farm, Muggleswick, Consett (Feb. 8); Aller Cleugh, Ireshopeburn, Wearhead, Bishop Auckland (Feb. 10).
Leics. Brook Farm, Saxelby, Melton Mowbray (Feb. 9).
Renfrew. Deval Farm, Port Glasgow (Feb. 8).
Yorks. Grange Farm, Wincobank Lane, Sheffield 23, Fitzwilliam Street, Swinton, Mexborough (Feb. 7); Warren House Farm, Swillington Common, Swillington, Leeds; Hartsoil Farm, Cleckheaton (Feb. 8); Birk House Farm, Cross Lanes, Barnard Castle; Old Hall Farm, Burstwick, Hull (Feb. 10).

Fowl Pest

Derby. Mill Farm, Millthorpe, Holmesfield (Feb. 9).
Essex. Homestead, North End, Dunmow (Feb. 9).
Lincs. The Chestnuts, Billingborough, Sleaford (Feb. 7); Stud Farm, Horbling, Sleaford (Feb. 9); 34, Grosvenor Road, Billingborough, Sleaford (Feb. 10).
Norfolk. The Heath, Heavingham, Norwich; West House Farm, Fressingfield, Diss (Feb. 10).
Surrey. Darenth and Dinkiddie, Frog Grove Lane, Wood Street, Guildford (Feb. 8); Kampar, Frog Grove Lane, Wood Street, Guildford (Feb. 10).

Swine Fever

Cambs. Springfield, Cherry Hinton, Cambridge (Feb. 9).
Ches. Pinfold Farm, Wybunbury, Nantwich (Feb. 7); Holtridge Farm, Norbury (Feb. 10).
Cornwall. Holwood, Saltash (Feb. 9).
Denbighs. Eyton House, Eyton, Nr. Wrexham (Feb. 14).
Essex. Maltbeggars Farm, Coggeshall, Colchester (Feb. 8); Ware Farm, High Roding, Dunmow; North Station Allotments, Colchester (Feb. 14).
Herts. Monkbury Farm, Little Hallingbury, Bishop's Stortford; Rushing Wells Farm, Barkway, Royston (Feb. 14).
Kent. 342, Hempstead Road, Gillingham; Home Street Farm, Frindsbury, Rochester (Feb. 7).
Lancs. Lucas Green Farm, Whittle-le-Woods, Chorley (Feb. 14).
Leics. Fields Farm, Lutterworth Road, Burbage, Hinckley (Feb. 10).
Lincs. Burrow's Farm, Lincoln Road, Dorrington (Feb. 7).
Monmouth. 125, High Cross Road, Rogerstone, Newport (Feb. 7); Mervale Farm, Watery Lane, Liswerry, Newport (Feb. 8); Little Green Court Farm, Bettws, Newport (Feb. 11).
Norfolk. Swamp Lane Farm, Great Ellingham, Attleborough; Manor Farm, Smallburgh, Norwich; Woodhaven, Thorpe Road, South Repps, Norwich (Feb. 7); Wensum Nurseries, Taversham Lane, Costessey, Norwich (Feb. 12).
Salop. Pool Farm, Osbaston, High Ercall, Wellington; Tern Hill Hall Farm, Tern Hill, Market Drayton (Feb. 10); Bridge Farm, Edstaston, Wem (Feb. 11); Sunnyside Farm, Aston, Wem (Feb. 14).
Staffs. Park Green Farm, Foxt, Stoke-on-Trent (Feb. 14).
Warwicks. Manor Road Piggeries, Four Ashes Road, Dorridge, Birmingham (Feb. 8).
Worcs. Goodyears Farm, Shrawley, Nr. Worcester (Feb. 14).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

GREEN YOLK EGGS

Sir,—The letter in your issue of January 29th from Mr. H. M. F. Maynard is in humorous vein, but two inferences to be drawn from it are (1) that the results of research relating to poultry husbandry are regarded as of little interest to members engaged in general practice; and that (2) the significance of "green yolks," despite the widespread publicity afforded to this term only a few years ago, is not fully understood.

The condition known variously as "green yolks," "olive yolks" and "grass eggs" is, as these names imply, characterised by an olive to grass-green discoloration of the yolk. It has been recognised for well over 30 years, and is clearly to be distinguished from the abnormal types of pigmentation that may result, in certain circumstances, from the feeding of acorns, cottonseed, peanuts, soya beans or linseed meal, although some of these may involve an olive appearance. The concentration of green pigment in the yolk itself may increase on storage, with consequently a greater tendency for the candler to downgrade the egg in question, but there is no relationship to bacterial decomposition or to "badness." The egg is perfectly palatable, and may be consumed without harm. It does not appear to differ from a normal egg in anything but colour, and its only objectionable feature lies in its unusual appearance.

In Great Britain there appeared in 1948 and 1949 to be a significant increase, at certain packing stations in particular, in the number of eggs downgraded on account of this condition. Many green yolk eggs reached the general public, and such was the attendant publicity that the front of one of the National weeklies bore a coloured illustration of a typical specimen. The majority of cases occurred in the produce of birds maintained under extensive conditions. Various earlier attempts to reproduce the condition had yielded negative or equivocal results, but Mr. Alan W. Davies, then of the Animal Health Trust, was able to do so in feeding experiments involving the use of grain/clover and grain/lucerne diets, or by dosing laying birds with a chlorophyll preparation. He was able also to obtain presumptive evidence that the responsible pigment in the yolk itself was a chlorophyll derivative. These results were reported to the IXth World's Poultry

Congress in 1951. In the following year, Dr. Dudley E. Eyles, of the Grassland Research Station, reported the occurrence of green yolks in the eggs of birds fed a diet containing extracted grass leaf protein.

It has generally been held that the oil-soluble chlorophyll present in the green plant is not absorbed from the alimentary tract, and decomposition products have been identified in the stomach walls of herbivora. The product demonstrated spectrophotometrically by us to have passed to the yolk was a water-soluble derivative: it is nevertheless of interest that, despite reports to the effect that products of this type are capable, when administered by mouth, of affecting the odour of sweat and vaginal secretions, there has, hitherto, been no direct demonstration of their absorption in any of the higher animals. The relationship of oil-soluble chlorophyll to "green yolks" is not yet clear, although a mechanism similar to that postulated by Drs. W. Bolton & R. H. Common (*Nature, Lond.*, 1941, **148.** 373), and dependent upon the *p*H of the crop contents, may be involved.

Yours faithfully,

ALASTAIR N. WORDEN,

Cromwell House,
Huntingdon.

January 31st, 1955.

THE TOXICITY OF STILBOESTROL

Sir,—The experiment reported by Taylor and Gordon (issue of January 15th, 1955) was part of an informally co-ordinated test carried out at five research centres. The results of the co-ordinated test will appear in the *British Journal of Nutrition*, Vol. 9, No. 2. Readers of the Taylor and Gordon paper will be well advised to reserve interpretation of the results until the co-ordinated report is published.

Yours faithfully,

R. BRAUDE,

National Institute for Research
In Dairying,
Shinfield,
Nr. Reading.

January 24th, 1955.

Readers will have noted that Messrs. Taylor and Gordon stated both in the body of their paper and in its addendum that the work described formed part of a co-ordinated trial the results of which would be published later. We are obliged to Dr. Braude for giving us in advance a reference to the published results of this co-ordinated trial.—Editor.

R.V.C. NEW FIELD STATION

Sir,—Towards the end of 1954 London students at the Royal Veterinary College were informed of the proposed move to Potters Bar during this year. This place has been chosen as most suited for the Final Year Field Station.

To my mind this is nothing less than a tragedy for the future Final Year students. Streatley was not a perfect example of a field station, but there were all the ingredients available to make it so. Research stations at Compton and Shinfield, Reading Agricultural College, the College practice at Wallingford, a very good support from local farmers, and plenty of opportunity to enjoy oneself in one's free time were notable advantages. Surely this could be developed into a fine field station?

Now, Sir, can someone please let me into the secret of the attraction of Potters Bar? Who is to benefit by such a move? Most certainly not the students. What is their opinion of this move? What is the opinion of those members who have qualified in Streatley on this subject?

Perhaps this letter may be regarded as being impolite to my previous tutors, but is it not time that some liaison existed between members and the various teaching colleges with regard to methods of instruction, or at least that there should be a chance to voice opinions on such major changes as the Potters Bar venture?

Yours faithfully,

MICHAEL STALBOW.

The Haven,
Earl Soham,
Woodbridge, Suffolk.

February 1st, 1955.

CANINE DISTEMPER AND CANINE ENCEPHALITIS

Sir,—During the course of recent investigations in the field concerning canine distemper and canine encephalitis, we have been faced with the difficult problem of advising on the best course of therapy, particularly in cases where nervous manifestations appear to be imminent. In these cases we have observed that the use of egg adapted virus appears to precipitate the onset of encephalitis.

As no specific line of treatment is yet available, resort must be made to existing therapeutic agents. An interesting observation arose as a result of field trials with immune gamma globulin. It was found that by obtaining a high level of antibody, over a period of 12 to 14 days, some very encouraging results were obtained.

Such levels were accomplished by using gamma globulin in a similar way to the antibiotics, *i.e.*, a dose equivalent to 10 ml. serum every 48 hours until six or seven doses had been administered. Recently, quite encouraging results have also been obtained by the use of therapeutic distemper serum at the rate of 0.5 ml. per lb. injected at the same intervals.

If scleral congestion suggests the presence of a subclinical encephalitis, we advise the use of Mysoline (I.C.I.), in conjunction with this therapy. This drug is commenced in small doses and over a period of seven days is gradually increased to the full dosage, which is maintained for long periods up to four to six weeks if necessary. At the end of the course, the drug is eased off as gradually as it was commenced.

It is important to make as early a diagnosis as possible, give the antibody as advised, and even repeat this dosage in two to three weeks, if any doubt

exists regarding recovery. Even in cases where the prognosis has been extremely grave, the result of this line of therapy has often been most satisfactory.

It is realised that such treatment is expensive, but by using these measures, particularly in the early course of the disease, it is possible that the duration of the illness is reduced considerably. No doubt others have used similar treatment, if so, the results obtained would be of considerable interest to us.

Yours faithfully,

S. F. J. HODGMAN,
W. B. SINGLETON.

Canine Research Station of the
Animal Health Trust,
Kennett,
Nr. Newmarket.

February 11th, 1955.

BOWEL OEDEMA

Sir,—An investigation is at present being carried out at this laboratory into the aetiology of bowel oedema. I am anxious to obtain samples of blood and urine from as many clinical cases as possible.

May I appeal through your journal to veterinary surgeons who encounter outbreaks of this disease to let me have samples of 1 to 5 c.c. of whole (uncitrated) blood and any amount of urine? Samples from dead animals are of no value. Veterinary surgeons living within about 50 miles of Langford are also invited to let me know of an outbreak as soon as possible so that someone from this laboratory may have an opportunity to collect samples from live animals. Acknowledgment will be duly recorded.

Yours faithfully,

R. J. GARNER.

University of Bristol Veterinary Laboratory,
Langford House,
Langford, nr. Bristol.

February 8th, 1955.

A LEGITIMATE GRIEVANCE

Sir,—It is certainly interesting to have the opinion of a layman on this subject, but I feel that one or two points raised by Mr. Turner deserve comment.

The fact that there are people who *could* pay for veterinary treatment but *will* not is largely the reason for this correspondence. Whether such people will not pay for treatment because of meanness or negligence, or both, they are all equally guilty of some degree of cruelty to their animals. Does Mr. Turner think that irregular or sporadic treatment by a travelling or other free clinic is likely to ensure that the unhappy animals belonging to such people are likely to receive kind treatment otherwise? It seems to me that such people are quite irresponsible and should never be allowed to keep a pet. To neglect an animal to such an extent as to cause suffering is an offence in law, and such people should be reported and prosecuted, and not pandered to by the mistaken efforts of welfare societies.

There are only two valid reasons for the existence of free animal clinics and welfare societies. The first is to treat the animals of the *genuinely* poor, and the second is to detect and prosecute people who are cruel to animals. An animal would be better dead than in the hands of a person who is so mean, negligent or dishonest as to refuse to spend a few shillings on its treatment when it is ill.

The private veterinary surgeon seldom has trouble in almoning. People who are genuinely in need and are really honest will not be asked many questions. In my own experience decent people who have struck bad times quite honestly ask how much treatment is likely to cost and explain their position. They always get full and competent treatment whatever their circumstances.

People who buy T.V. sets to keep up appearances do not require to keep a pet they cannot afford to treat decently, and should make up their minds which they prefer. People who have cars supplied by their firms are usually in a reasonably good position and could surely afford reasonable treatment for their animals.

Yours faithfully,

MILLER WATSON.

11, Annfield Place,
Glasgow, E.1.

February 6th, 1955.

Sir,—I have read with interest the layman's point of view as put forward by Mr. Turner in your recent issue. This touches on the point in my original letter to you: these people to whom he refers should not be encouraged to keep animals as pets.

The proper way to deal with this problem is not, I believe, the establishment of more free clinics, it is for public spirited persons such as Mr. Turner to report these callous dog-owners to the R.S.P.C.A., who will then take action against them, for cruelty in neglecting to give their pets' ailments proper attention.

Yours faithfully,

LUCY R. ROBINSON.

The Grey House,
Purleigh,
Chelmsford, Essex.

February 11th, 1955.

MYXOMATOSIS

Sir,—When I worked in the Lake District a large percentage of the working dogs were fed a diet mostly of flaked maize and milk. Cases of nicotinic acid deficiency were common—in spite of an abundance of rabbits—and responded dramatically to large doses of nicotinic acid. This, Sir, was in 1949 when myxomatosis was of entirely academic interest in Great Britain.

Yours faithfully,

OLAF SWARBRICK.

Surrey House,
Arundel, Sussex.

January 30th, 1955.